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Air Pollution Assessment of Trichloroethylene

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Air Pollution Assessment of Trichloroethylene

B.B. FULLER

FEBRUARY 1976

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Trichloroethylene is an organic solvent primarily used for the vapor degreasing of metals. Approximately 200,000 industrial workers are exposed to this solvent annually. Although the major physiological response in humans from acute or chronic exposure to trichloroethylene is central nervous system depression, damage to liver, kidney and heart have also been reported. Since the metabolic fate and toxic effects of trichloroethylene are similar in many mammalian species to those in man, the fact that this compound has recently been implicated as a potent liver carcinogen in mice may be of significance.

Approximately 60% of the total world production of trichloroethylene is released to the environment each year. However, due to its low water solubility, high vapor pressure and high atmospheric photodegradation rate, trichloroethylene is not expected to persist in the environment. Ambient concentrations in the atmosphere of industrialized areas are only about 18 ppt. Proper use of local exhaust systems in conjunction with vapor condensation appratus and good general ventilation should be sufficient to maintain levels of trichloroethylene in the workshop environment well below the recommended 100 ppm and to insure a minimum release to the ambient atmosphere.

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AIR POLLUTION ASSESSMENT OF TRICHLOROETHYLENE

I. SUMMARY AND CONCLUSIONS

Trichloroethylene is a noncorrosive, nonflammable liquid with excellent solvent properties. The compound is easily oxidized to products which are both acidic and corrosive. Oxidation inhibitors are available which are effective at concentrations of less than one percent.

Over 90 percent of all the trichloroethylene produced is used by industrial metal fabricating plants for vapor degreasing. The remainder is utilized as an extractive solvent in foods and medicines and as a dry cleaning solvent. Trichloroethylene was also the solvent used to extract caffein from coffee, and was present to the extent of 10 to 25 ppm. General Foods, Inc., the largest manufacturer of decaffeinated coffee, has recently replaced trichloroethylene with methylene chloride.

The major physiological response in humans from acute or chronic exposure to trichoroethylene is central nervous system depression. This response may take the form of visual disturbances, mental confusion, fatigue, nausea and vomiting, dizziness, palpitation, and other symptoms. These effects have actually been observed in industrial workers exposed to trichloroethylene vapors as well as in experimental subjects. Evidence indicates that adverse effects are noticeable in humans even at the allowable level of 100 ppm. Mild

effects have even been reported at concentrations as low as 5 to 10 ppm. One-hundred-and-four workers were exposed to this chemical for over 3 years at levels of about 100 ppm. Ninety-two percent of these workers exhibited some of the symptoms described above. However, cessation of exposure reversed the effects. Symptoms subsided in most of the workers within five months. Data relating to this type of effect are, in general, quite conclusive. Additional effects such as, damage to liver, kidney, and heart have also been reported. These data, in many cases, are clouded by the fact that the subjects were also heavy consumers of alcohol. This factor may have contributed to or aggravated the observed condition. Therefore, evidence for this type of damage is inconclusive. Blindness, loss of hearing and tactile sense, and death from cardiac arrest and ventricular fibrillation following acute exposure have also been reported.

The major products of trichloroethylene metabolism are trichloroethanol and trichloroacetic acid. Trichloroethanol is much more toxic to the heart and nervous system than trichloroethylene. Thus, it is believed that this metabolite is responsible for many of the observed toxic effects. High levels of trichloroethanol are excreted by persons exposed to as little as 70 ppm of trichloroethylene.

Metabolic fate and toxic effects of trichloroethylene in many mammalian species are similar to those in man. Moderate or reversible liver damage was reported in mice, guinea pigs, and dogs

following inhalation of concentrations 10 to 16 times the acceptable industrial level (100 ppm). Inhalation at levels of 2 to 20 ppm led to inhibition of antibody formation and of neutrophil activity. Cats exposed to 20 ppm for 60 to 90 minutes for 4 to 6 months showed damage to liver, kidneys, spleen, and lymph nodes. Concentrations of 7000 to 14000 ppm (70 to 140 times that of industrial exposure) produce changes in the electroencephalogram in rabbits, in some cases resembling epileptic seizures. The applicability of these experimental results to humans is uncertain. Mice are especially sensitive to hepatotoxic agents and rabbits, to convulsions.

Trichloroethylene has recently been implicated as a potent liver carcinogen in mice. It was administered by gastric intubation at massive dose levels (900 to 2400 mg/kg), and a maximun of 43.2 percent of the test animals were affected. This claim for carcinogenicity is not entirely conclusive. Effects produced in highly susceptible animals (2.5 percent of the controls spontaneously developed liver cancer) could not be repeated in rats. In any event, dosage levels are equivalent to a human drinking 50 million cups of decaffeinated coffee each day for a lifetime.

Inhalation studies conducted at the laboratories of Dow Chemical Corporation showed that trichloroethylene was not teratogenic in mice or rats at 300 ppm. No mutagenesis studies have been reported.

Trichloroethylene is not expected to accumulate in the environment because it is slightly soluble in water, possesses a high vapor

pressure, and has a high atmospheric photodegradation rate (a half-life of 0.3 days at sea level). Therefore, it is not felt to represent an air pollution hazard. Ambient concentrations in the atmosphere of industrialized areas are about 16 parts per trillion; in rural areas, they are less than 5 parts per trillion; ambient concentrations in water are about 0.6 nanograms/liter (0.1 ppt)

Ground-level concentration of trichloroethylene in the atmosphere 500 meters downwind of a production plant is estimated to be approximately 570 g/m^3, or 0.106 ppm. Assuming that symptoms occur following chronic industrial exposure to 5 ppm and that the average industrial worker is exposed for 2,000 hours per year, an individual must remain at this point 24 hours a day for 11.4 years in order to inhale a similar amount. He must remain there for 228 years in order to inhale an amount equivalent to that inhaled by an industrial worker following a 1-year exposure to 100 ppm.

The most significant population exposed to trichloroethylene is industrial workers involved in vapor degreasing or dry cleaning operations. About 200,000 such workers are exposed each year. In 1973, OSHA recommended an ambient level of 100 ppm as the allowable time-weighted average concentration over an 8-hour workday, with a 300 ppm excursion limit. Evidence suggests that excursions considerably above this level may exist. The general public is exposed to trichloroethylene through inhalation of cleaning fluids and ingestion of foods, spices, and medicines. Ingestion exposure is a result of

consuming products whose commercial preparation required an extraction operation with this solvent. Residue concentrations ranging from 0.02 to 22 µg/kg have been detected in foodstuffs and concentrations of up to 32 µg/kg have been detected in human tissue. Trichloroethylene is rapidly absorbed by the lungs following inhalation, but only slightly eliminated upon exhalation.

Current measurement technology is adequate to ensure that workers are not exposed to hazardous levels. Adsorption onto activated charcoal represents the easiest and most efficient method of collection, and gas chromatography is the most specific and sensitive method for analysis. Breath analysis of exposed workers is also feasible.

Trichloroethylene is a synthetic chemical manufactured both domestically and abroad. Estimated world annual production in 1973 was approximately one million tons and domestic production in 1974 amounted to about 215,000 tons. There are five producers of trichloroethylene in the United States and their production plants are all located in southern Texas and Louisiana.

About 600,000 tons of trichloroethylene are released to the atmosphere and 10,000 tons to the ocean each year. This total release represents approximately 60 percent of total world production.

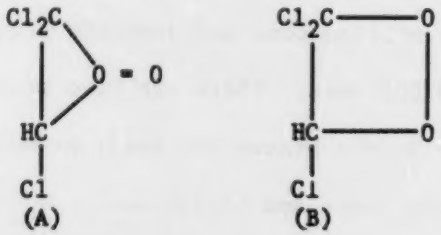
A proper local exhaust system, vapor condensation apparatus, and general ventilation system should keep levels below 100 ppm. Care must be taken to clean up spills, check leaks, and repair faulty equipment.

II. AIR POLLUTION ASSESSMENT REPORT

A. PHYSICAL AND CHEMICAL PROPERTIES

Trichloroethylene, $CHCl = CCl_2$, is at normal temperatures a clear, colorless, noncorrosive, nonflammable liquid, possessing the characteristically sweet odor associated with chlorinated hydrocarbons. Physical properties are listed in Table I (1, 2, 3).

Trichloroethylene is unstable in the presence of oxygen. Degradation is further promoted by elevated temperatures and exposure to light (especially ultraviolet irradiation). It is quite reactive to OH, but only very slowly oxidized by alkyl peroxy radicals and ozone (4). Over 80 percent of the initial oxidation products (as indicated below) consist of compound (A), which decomposes to yield dichloroacetyl chloride, $Cl_2CHCOCl$ (2).



(A) (B)

Compound (B), another initial oxidation product, yields phosgene, carbon monoxide, and hydrogen chloride upon further decomposition. In the presence of anhydrous aluminum chloride, compound (A) rearranges to yield equal amounts of chloroacetyl chloride and chloral, CCl_3CHO. Compounds (A) and (B) are violently explosive and their sudden decomposition is promoted by the addition of impurities, including oxidation

TABLE I

PHYSICAL PROPERTIES

Molecular formula:	$ClCH = CCl_2$
Physical state:	colorless liquid
Molecular weight:	131.4
Melting point, °C:	-87.1
Boiling point 760mmHg, °C:	86.7
Specific gravity	
20°/4°C:	1.464
25°/4°C:	1.456
100°/4°C:	1.325
Vapor density, bp, 1 atm g/l (air = 1):	4.54

Vapor pressure:	°C	mmHg	°C	mmHg
	-20	5.4	30	94
	-10.8	10.8	40	146.8
	0	20.1	50	212
	10	35.2	60	305.7
	20	57.8	86.7	760

Viscosity, liquid:	°C	CP*	°C	CP*
	-80	2.95	0	0.7
	-70	2.10	20	0.58
	-60	1.68	40	0.48
	-30	1.02	60	0.42
	-20	0.9		

*centipoise

TABLE I (CONCLUDED)

Solubility of trichloroethylene in water, g/100g H_2O	
25°C:	0.11
60°C:	0.125
Solubility of water in trichloroethylene g/100 g trichloroethylene	
0°C:	0.01
25°C:	0.033
60°C:	0.080
Percent in "saturated" air (25°C):	10.2
Density of "Saturated" air (air = 1):	1.35
Ignition temperature, °C:	463
Flash point:	none by standard methods
Refractive index (20°C):	1.478
Dielectric constant (liquid) 16°C:	3.42
Odor threshold (ppm)	21.4
Conversions:	1 ppm = 5.38 mg/m^3 @25°C
	1 mg/liter = 186.1 ppm

inhibitors. In the presence of moisture, dichloroacetyl chloride is decomposed to dichloroacetic and hydrochloric acids. The acidic products of oxidation are highly corrosive. They react with most metals with which the liquid may contact.

A large variety of pure compounds and mixtures are effective oxidation inhibitors of trichloroethylene. Normally, these stabilizers are effective at less than one percent by weight. Some current stabilizers in use include: acetone, acetylenic compounds, aniline, borate esters, n-butane, O-cresol, diisopropylamine, ethyl acetate, hydrazine derivatives, isobutyl alcohol, lactones, O-nitrophenol, pyrazoles, stearates, and SO_2. With inhibitor addition, trichloroethylene is stable (to 130°C) in the presence of air, moisture, light, and common construction metals. Above 130°C, inhibitors are ineffective and corrosion of the metals occurs. When heated above 700°C, the vapor decomposes, yielding a mixture of dichloroethylene, perchloroethylene, carbon tetrachloride, chloroform, and methyl chloride. When heated vigorously in the presence of air, trichloroethylene vapor is completely oxidized to carbon dioxide and hydrogen chloride. Reaction with ozone yields an explosive ozonide which decomposes to hydrogen chloride, phosgene, carbon monoxide, and chlorine peroxide (2).

Trichloroethylene is not readily hydrolyzed by water. Under pressure at 150°C, it reacts with alkaline hydrazides to yield glycolic acid, $CH_2OHCOOH$. Reaction with 90 percent sulfuric acid yields

monochloroacetic acid. Strong alkalis dehydrochlorinate trichloroethylene, with the production of explosive and flammable chloroacetylene (2).

B. EFFECTS

1. Humans. Trichloroethylene is rapidly absorbed by the lungs upon inhalation. However, only slight amounts are eliminated upon subsequent exhalation. An early indication of the metabolic fate of absorbed trichloroethylene was revealed in a study by Barrett and Johnston (5). In this effort, the steam distillate of urine from human subjects exposed to trichloroethylene was found to contain a metabolite with three chlorines on a single carbon atom. Further investigation in dogs led to the conclusion that this metabolite was trichloroacetic acid. This conclusion was confirmed by Powell in 1942 (6). A somewhat later study by Butler using dogs (7) revealed that the major product of trichloroethylene metabolism was trichloroethanol, either free or conjugated with glucuronic acid. Butler concluded that the metabolism of trichloroethylene involves an initial conversion to chloral hydrate with rapid metabolic conversion to either trichloroethanol or trichloroacetic acid.

In 1960, a detailed quantitative study was performed by Soucek and Vlachova (8). They showed that an average of 64 percent (range, 58 to 70 percent) of inhaled trichloroethylene vapor was retained by persons exposed to concentrations of up to 150 ppm.* Within a few minutes of exposure, monochloroacetic acid (a minor metabolite), trichloroacetic acid, and trichloroethanol were accumulating in the

---

*Duration of exposure was not reported.

urine. The excretion of monochloroacetic acid was maximal at the end of exposure and continued for as long as 168 hours after exposure. Monochloroacetic acid constituted about four percent of the retained trichloroethylene. The excretion of trichloroacetic acid was maximal 24 to 28 hours following exposure and continued for as long as 520 hours. Trichloroacetic acid constituted from 10 to 30 percent of the retained trichloroethylene. Finally, excretion of trichloroethanol was maximal a few hours following exposure and continued for as long as 216 hours. Trichloroethanol constituted from 32 to 59 percent of the retained trichloroethylene. The total amount of metabolites excreted amounted to 43 to 100 percent of the absorbed trichloroethylene and were in the ratio monochloroacetic acid: trichloroacetic acid: trichloroethanol::1:5:12. Later studies by Bartonicek (9) and Ogata et al. (10) generally confirmed the results outlined above. Analyses of postmortem human tissue revealed the presence of trichloroethylene in body fat, kidney, liver, and brain, indicating uptake by these tissues. Concentrations in eight subjects are presented in Table II.

In 1972, Ikeda et al. (11) indicated that the high percentage of trichloroethanol excreted by persons exposed to quantities of trichloroethylene in excess of 70 ppm might have serious toxicological consequences since trichloroethanol is much more neurotoxic* and cardiotoxic** than trichloroethylene itself. These results suggest

*Poisonous or destructive to nerve tissue.
**Having a poisonous or deleterious effect upon the heart.

TABLE II

OCCURRENCE OF TRICHLOROETHYLENE IN HUMAN TISSUE.
CONCENTRATIONS IN μg/kg (WET TISSUE)

AGE OF SUBJECT	SEX	TISSUE	TRICHLORO-ETHYLENE
76	F	Body Fat	32
		Kidney	< 1
		Liver	5
		Brain	1
76	F	Body Fat	2
		Kidney	3
		Liver	2
		Brain	< 1
82	F	Body Fat	1.4
		Liver	3.2
48	M	Body Fat	6.4
		Liver	3.5
65	M	Body Fat	3.4
		Liver	5.2
75	M	Body Fat	14.1
		Liver	5.8
66	M	Body Fat	4.6
74	F	Body Fat	4.9

Source: McConnell, G., Ferguson, D.M., and Pearson, D.R. "Chlorinated Hydrocarbons and the Environment" Endeavor 34 13-18 (1975).

that the standards proposed by the Occupational Safety and Health Administration (100 ppm allowable time-weighted average concentration over an 8-hour workday with a 300 ppm limit) may not be adequate.

Many studies of the effects of chronic occupational exposure to trichloroethylene have been reported. In general, a wide spectrum of complaints were observed. A study involving 70 young workers (83 percent less than 30 years old) exposed up to 6 years to variable concentrations of trichloroethylene reported the following symptoms (12): headache (74 percent), dizziness (31 percent), nausea (43 percent), sleepiness at end of shift (29 percent), fatigue (68 percent), euphoria (31 percent), palpitations (29 percent), disturbances of vision (21 percent), irritability (56 percent), disturbed sleep (46 percent), anxiety (27 percent), loss of appetite (50 percent), excessive sweating (39 percent), and alcohol intolerance (21 percent). The following additional symptoms were reported in studies by Andersson (13) and Bardodej and Vyskocil (14): vertigo, tremors,* vomiting, a feeling and appearance of light-headedness or "drunkenness," and slowing of the heartbeat (bradycardia). Andersson reported that only 8 of 104 workers who had been exposed to this chemical for more than 3 years were without symptoms. However, cessation of exposure reversed the effects and follow-up studies three to seven years later showed little residual evidence of trichloroethylene

*A hallucination of movement, a sensation as if the external world were revolving around the subject or as if he himself were revolving in space.

intoxication. Some workers reported that symptoms had subsided within five months after termination of exposure. Effects on skin included reddening and dermographism,* skin burns on contact, generalized dermatitis resulting from contact with vapor, and possibly scleroderma** (15, 16, 17). Stewart and Dodd (18) demonstrated that unless trichloroethylene is trapped against the skin, absorption by this route is insignificant.

In one study (19), an attempt was made to correlate levels of trichloroacetic acid excreted in the urine of 122 exposed workers with the results of an independent medical survey. The correlations were as follows: less than 20 mg trichloroacetic acid per liter of urine, no symptoms; 40 to 75 mg, 50 percent reported abnormal fatigue, increased need for sleep, diffuse gastric symptoms, irritability, headache, and intolerance of alcohol; 100 mg to 200 mg, most workers reported some of the above symptoms; greater than 200 mg, most workers reported symptoms plus an increased absence from work because of illness; greater than 300 mg, all of the workers experienced all of the above symptoms. Andersson (13), in a detailed study of 104 exposed workers confirmed this report, indicating that no symptoms appeared when excretion of trichloroacetic acid was less

*A condition in which scratching the skin with a dull instrument provokes a linear, raised, pale streak.

**A systemic disease which may involve the connective tissues of any part of the body, including the skin, heart, esophagus, kidney and lung. The skin may be thickened, hard and rigid and pigmented patches may occur.

than 20 mg/liter of urine, but that most workers reported symptoms when excretion was greater than 75 mg/liter.

Frant and Westendorp (20) calculated that a person subjected to a sustained exposure of 100 ppm for several days will excrete about 200 mg of trichloroacetic acid/liter of urine. Fribert el al. (21) confirmed this estimate by exposing 3 persons for 7 hours daily for one week to 100 to 150 ppm and noting concentrations of 250 to 300 mg trichloroacetic acid/liter of urine during the latter days of the study. Thus, some idea of the levels to which the above workers were exposed is possible.

In another study (14), Bardodej and Vyskocil examined 75 workers exposed to trichloroethylene in dry cleaning and degreasing operations for periods of from less than one year to ten or more years. In general, symptoms increased with length of exposure. These investigators also attempted to correlate symptoms with levels of exposure. The results are summarized in Table III. Cases of chronic overexposure resulting in total blindness (22, 23) and in total loss of tactile* sense and inability to grasp objects between thumb and forefinger (16) have been reported. Physiologic changes observed following chronic exposure include increased levels of leukocyte alkaline phosphatase and blood acid phosphatase (24). It is suggested that these findings might indicate an increased capacity to metabolize

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*Pertaining to the touch.

TABLE III

PERCENT OF WORKERS EXHIBITING EaCH SYMPTOM AT THE GIVEN EXPOSURE LEVEL

	30-630 ppm	100-154 ppm	5-10 ppm
Headache	70	26	67
Intolerance to alcohol	92	63	22
Disturbance of sleep	83	26	22
Fatigue	92	47	61
Bradycardia	33	40	16
Electrocardiogram Disturbance	16	21	6

Source: Bardodej, Z., and Vyskocil, J. "The problem of trichloroethylene in occupational medicine" Arch. Ind. Health 13, 581-592, 1956.

alcohols; represent a response to changes in blood pH; or might be related to glycogen metabolism in the liver. Exposure to trichloroethylene may either speed or slow the heart rate. Andersson (13) reported abnormal electrocardiograms with disturbance of cardiac rhythm in 77 out of 104 exposed workers studied.

To what extent trichloroethylene damages the liver is not clear. Albahary et al. (25) and Talot et al. (26) conducted liver function tests on exposed workers and reported no evidence of liver disorders. Milby (27) noted normal liver and kidney function in a severely overexposed worker who was excreting 780 mg trichloroacetic acid per liter of urine. On the other hand, Guyotjeannin and Van Steenkiste (28) reported abnormalities of cephalin flocculation,* total lipids, and unsaturated fatty acids and an increase in beta and gamma globulins in 18 regularly exposed workers.

Laboratory studies on human volunteers have been conducted in order to elucidate the effect of exposure to trichloroethylene on performance in various psychophysiological tests. Vernon and Ferguson (29) subjected 8 male volunteers to two-hour exposures of 0, 100, 300, and 1000 ppm of trichloroethylene. Decrements in performance were significant only at 1000 ppm. In a later study, Stewart

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*Cephalin flocculation test (Hanger's test) is a test for the presence of liver cell disease, based on the flocculation of a cephalin-cholesterol emulsion by the patient's serum. Cephalin is a crude phospholipid which is used as a clotting agent in blood coagulation work.

et al. (30) exposed 5 subjects to 200 ppm for 7 hours on 5 consecutive days and reported normal performance on tests. On the other hand, Salvini et al. (31) exposed 6 male volunteers to an average concentration of trichloroethylene of 110 ppm (range 90 to 130 ppm) in two 4-hour sessions separated by 1.5 hours. He reported a significant decrement in performance on tests of memory, perception, and manual dexterity and concluded that 100 ppm was very close to the average concentration capable of interfering with psychophysiological efficiency. These studies would appear to support those of Ikeda et al. (11), again suggesting that present occupational exposure levels may be unhealthy.

Cases of acute exposure with extremely serious and often fatal consequences have been reported. Toxic action primarily involves the central nervous system, with specific paralysis of the trigeminal nerve,* but the gastrointestinal system, circulatory system, and occasionally the kidneys are affected as well.

The first report of deaths resulting from acute overexposure in the United States was presented by Kleinfeld and Tabershaw (32). In one case, the victim died of apparent hepatorenal** failure after accidentally drinking trichloroethylene. The man had, however, been a heavy drinker, which might have been a contributing factor. Secchi

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*The fifth cranial nerve.
**Pertaining to the liver and kidneys.

et al. (33) reported that no liver damage was observed when pure trichloroethylene was ingested. Four other victims were all employed at degreasing operations where concentrations of trichloroethlene approached 8000 ppm. All four complained of nausea, drowsiness, dizziness, and vomiting and died suddenly within a few hours after leaving work. The cause of death was ventricular fibrillation.*

In 1963, James (30) reported the case of a man employed as a vapor degreaser who had become addicted to trichloroethylene. He exhibited all of the usual symptoms associated with chronic exposure but also lost his sense of smell. He died suddenly of cardiac arrest 17 hours after his last known exposure. Autopsy also revealed fatty degeneration of the liver.

Other cases of sudden death occurring within a short time after acute exposure to trichloroethylene have been reported (31-33). In one case, death was preceded by the usual symptoms and also paralysis of the face and neck muscles. In another case, the cause of death was ventricular fibrillation and, in the third case, massive liver necrosis** was observed. Lilis et al. (12) offered a theory that changes in epinephrine secretion associated with hypersympathicotonia*** induced by trichloroethylene exposure, particularly when

*A condition characterized by twitching of the ventricular muscle, the impulses traversing the ventricles so rapidly that coordinated contractions cannot occur.

**Death of tissue, usually as individual cells, groups of cells, or in small localized areas.

***An increased tone of the sympathetic nervous system.

accompanied by physical exertion or stress, might account for the cases of unexplained or sudden death.

Other effects resulting from acute exposure to trichloroethylene include loss of taste and trigeminal paralysis (38), complete loss of hearing for tones over 1000 cycles per second (39), and temporary paranoid psychosis with distortion of both vertical and horizontal vision (40).

No evidence for carcinogenicity of trichloroethylene in humans has been reported to date.

2. Animals. The metabolic fate of trichloroethylene is qualitatively the same in many mammalian species as it is in man. However, in some cases, there are quantitative differences. Rabbits, for instance, were shown to excrete 10 times more trichloroethanol than trichloroacetic acid and about 50 times less trichloroacetic acid than man (41). Rats, just as humans, excreted increasing amounts of trichloroacetic acid, with maximum excretion at 24 hours after exposure to 640 ppm, 1150 ppm, or 2500 ppm for 4 hours. They continued to excrete trichloroacetic acid for up to 144 hours (21).

Fabre and Truhaut (42) studied tissue homogenates from animals following exposure to trichloroethylene. They found trichloroethylene present in all of the tissues examined. Trichloroacetic acid was present in greatest concentrations in spleen, suprarenal glands, reproductive organs, and urine. In vitro metabolic studies concluded that the most active conversion of trichloroethylene to its metabo-

lites occurred in the spleen, followed by lung, brain, liver, and kidney.

Toxic effects following inhalation have been observed in a variety of species under both chronic (low concentration for long period of exposure) and acute (high concentration for short period of exposure) conditions. The highest levels of exposure with no observable effects on rats were: 0.3 hours at 20,000 ppm; 0.6 hours at 12,000 ppm; 1.4 hours at 4800 ppm; and 5 hours at 3000 ppm. The "no effect" levels for chronic exposure (7 hours a day, 5 days a week) were: 400 ppm, monkeys;200 ppm, rats and rabbits; and 100 ppm, guinea pigs (43). Prendergast et al. (44) demonstrated no effects levels in rats, guinea pigs, rabbits, dogs, and monkeys to be 730 ppm, 8 hours daily, 5 days per week, for 6 weeks and 36 ppm, 24 hours a day, for 90 days.

The wide range of adverse effects arising from chronic exposure to trichloroethylene can be seen from the study of Cagianelli et al. (45). In this effort, male rats were sprayed twice a day with this substance for 40 days. By the 40th day, some damage to the liver and heart and more serious damage to the kidneys was noted. Transaminase and γ globulin levels were increased and serum albumin levels were decreased. The experimental animals showed a marked decrease in weight and a reduction in mature red cells, reticulocytes,* leuko-

*A young red blood cell.

cytes,* and platelets** in the peripheral blood. Bone marrow activity was greatly depressed. Rats injected with 0.004 moles trichloroethylene per kilogram showed evidence of functional hepatic changes and microscopic evidence of liver damage 12 to 16 hours following exposure. These effects were apparently reversible and disappeared after 24 hours (46). Moderate liver damage was reported in guinea pigs exposed for over 1100 hours to levels of 1200 ppm trichloroethylene (47); in dogs exposed to 750 ppm repeatedly for 3 weeks (48); and in mice exposed to 1600 ppm, 4 hours a day, 6 times per week.

Cats exposed to 20 ppm trichloroethylene for 60 to 90 minutes for 4 to 6 months showed damage to liver, kidneys, spleen, and lymphatic ganglions*** (50), lending additional support to the Ikeda hypothesis (11).

Rats and rabbits exposed to trichloroethylene for 40 to 50 minutes, 5 to 6 times per week, showed levels of glutamic-oxalacetic transaminase and glutamic-pyruvic transaminase increased above normal by 58 percent and 27 percent, respectively (51). An intramuscular injection of a total of 8.75 to 9 mg/kg trichloroethylene in rabbits resulted in a 26 percent decrease in the level of ATP**** in the retina.

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*White blood cells.
**A circular or oval disk found in the blood of all mammals which is concerned in coagulation of the blood and contraction of the clot.
***Lymph nodes.
****Adenosine triphosphate.

Levels of aldolase, glucose-6-phosphate dehydrogenase, lactic dehydrogenase, and hexokinase were also decreased, whereas malate dehydrogenase and NAD* -sorbitol dehydrogenase were elevated. Most of the changes in enzyme activity were reversible; however, aldolase and glucose-6-phosphate dehydrogenase activity as well as ATP concentration failed to return to normal levels by two weeks following exposure (52).

Effects on the immune system were reported in rabbits exposed to 10 mg/m^3, 4 hours daily, for 9 weeks. These effects consisted of inhibition of antibody formation and of the activity of neutrophils** (53). Exposure of rabbits to 100 mg/m^3, 3 hours daily, for 8 to 10 months, resulted in a decreased total antibody titer following typhoid immunization.

Bradycardia was induced in guinea pigs by intraperitoneal injection of either trichloroethylene or its metabolite, trichloroethanol. The latter proved twice as effective in slowing the heart and the authors suggested that metabolic conversion to trichloroethanol was responsible for many of the observed toxic effects of trichloroethylene (55).

Effects on the brain and behavioral abnormalities have also been reported. Acute exposure of rabbits to 7000 ppm to 14,000 ppm for 15

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*Nicotinamide-adenine-dinucleotide.
**A white blood cell stainable by neutral dyes.

to 60 minutes produced changes in the electroencephalogram ranging from minor up to electroclinical epileptic seizures (56). Further studies on rabbits that were chronically intoxicated with alcohol revealed that subsequent exposure to trichloroethylene induced graver functional disturbances of longer duration even though the dose of trichloroethylene was half the strength used previously (57). These studies constitute evidence for the suggestion that alcohol may aggravate the effects of trichloroethylene intoxication. Rats exposed to 400 ppm for 8 hours, 5 times per week, exhibited decreased performance in swimming tests and increased exploratory behavior (58). Exposure to 800 ppm and 1600 ppm produced further decrements in performance with the animals exhibiting 65 percent and 58 percent of their normal activity, respectively (59).

The lethal concentration via inhalation for rats exposed for 4 hours was 8000 ppm (60). The lethal concentration in rabbits exposed for 50 minutes was 11,000 ppm (61). The concentration lethal to 50 percent of flatfish exposed for 96 hours (96 hours LC_{50}) was 16 mg/1 (2).

Trichloroethylene has recently been implicated as a potent liver carcinogen* in mice by the National Cancer Institute. Striking differences were observed between the incidence of hepatocellular

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*A cancer-producing substance.

carcinomas* in mice given trichloroethylene and in controls. Male mice were given either 2400 mg/kg or 1200 mg/kg doses, while females received 1800 mg/kg or 900 mg/kg. Doses were given by gastric intubation. Of the mice given the lower doses, 30.6 percent developed liver cancer. Of those receiving the higher doses, 43.2 percent were affected. The incidence of spontaneous liver cancer in the control group was 2.5 percent (63). Rats given a 500 mg/kg or 1000 mg/kg dose failed to develop cancer, but it was suggested that the extremely high dose was very toxic to the animals and might have clouded the study. Inhalation studies conducted at Dow laboratories, showed that trichloroethylene was not teratogenic** in mice or rats at 300 ppm (64). No mutagenesis studies have been reported. Toxicologic effects in all species, including man, are presented in Table IV.

Of all the effects reported in both animals and man, only those relating to central nervous system depression seem entirely conclusive. These effects, all of which are summarized in Table II, apparently occur at concentrations to which certain industrial workers may realistically be exposed and have, in fact, been shown to occur in such workers. Evidence on damage to the liver is not conclusive. "Moderate" or "reversible" damage has been demonstrated in animals at relatively high concentrations (10 to 16 times the typical industrial concentrations). The extent to which trichloroethylene damages the

*A malignant new growth made up of epithelial cells tending to infiltrate the surrounding tissues and spread.
**The production of physical defects in offspring in utero.

TABLE IV
TOXICOLOGICAL EFFECTS

ANIMAL	ROUTE	DOSE	LENGTH OF EXPOSURE	EFFECTS	REF.
Rat	Spray	—	2 times/day; 40 days	damage to liver, heart, kidneys loss of weight pancytopenia bone marrow depression changes in enzyme activities	45
	Injection	530 ppm	16 hours	reversible liver damage	46
	Inhalation	400 ppm 800 ppm 1600 ppm	8 hours; 5 times/week	progressive loss of activity	59
Mouse	Gastric intubation	900 - 2400 ppm	---	liver cancer	63
	Inhalation	1600 ppm	4 hours/day 6 days/week	liver damage	49
Guinea pig	Inhalation	1200 ppm	1100 hours	liver damage	47
	Injection	varied	---	bradycardia	55
Dog	Inhalation	750 ppm	3 weeks	liver damage	48

TABLE IV (CONTINUED)

ANIMAL	ROUTE	DOSE	LENGTH OF EXPOSURE	EFFECTS	REF.
Cat	Inhalation	20 ppm	60-90 minutes 4-6 months	damage to liver, spleen, kidneys and lymphatic ganglions	50
Rabbit	Inhalation	Varied	40-50 minutes 5-6 times/week	increased transaminase levels	51
	Injection	9 ppm	---	decreased ATP level changes in enzyme activity	52
	Inhalation	10 ppm	4 hours/day 9 weeks	inhibition of antibody formation and neutrophil activity	53
	Inhalation	100 ppm	3 hours/day 8-10 months	decreased total antibody titre	54
	Inhalation	7000 - 14,000 ppm	15-60 minutes	minor to major changes in electroencephalogram which are aggrevated by alcohol	56 57

TABLE IV (CONTINUED)

ANIMAL	ROUTE	DOSE	LENGTH OF EXPOSURE	EFFECTS	REF.
Human	Inhalation	1000 ppm	2 hours	decreased performance in psychophysiological tests	29
	Inhalation	90-130 ppm	two 4-hour sessions separated by 1.5 hours	decreased performance on tests of memory, perception and manual dexterity	31
	Oral	---	---	death from hepatorenal failure	32
	Inhalation	up to 8000 ppm	chronic	death from ventricular fibrillation	32
	Inhalation	varied	chronic	lost sense of smell, death from cardiac arrest; fatty degeneration of liver	34
	Inhalation	varied	acute	paralysis of face, neck muscles; death	35
	Inhalation	varied	acute	loss of taste; trigeminal nerve paralysis	38
	Inhalation	varied	acute	complete loss of hearing for tones over 1000 cps	39
	Inhalation	varied	acute	temporary paranoid psychosis; distortion of horizontal, vertical vision	40

TABLE IV (CONCLUDED)

ANIMAL	ROUTE	DOSE	LENGTH OF EXPOSURE	EFFECTS	
Human	Inhalation	varied (>20 ppm)	3-6 years	headache, dizziness, nausea, sleepiness, fatigue, euphoria, palpitation, disturbance of vision, irritability, disturbed sleep, anxiety, loss of appetite, excessive sweating, alcohol intolerance, vertigo, tremors, vomiting, bradycardia, other electroencephalograph disturbances	12 13 14 19
	Inhalation	varied	chronic	total blindness	22 23
	Inhalation	varied	chronic	total loss of tactile sense	16
	Inhalation	varied	chronic	increased levels of leucocyte alkaline phosphatase and blood acid phosphatase	24
	Inhalation	varied	chronic	abnormalities of cephalin flocculation, total lipids and unsaturated fatty acids, increase in beta and gamma globulins	28

*An abnormal or exaggerated sense of well-being.

human liver is difficult to ascertain since many of the subjects were heavy users of alcohol and this fact tended to cloud the studies (see, for example, 32).

The claim for carcinogenisis also is inconclusive. Studies were done using massive doses in highly susceptible animals (mice with a 2.5 percent incidence of liver cancer in the controls). In less susceptible animals (rats), no effects were observed. In any event, the dose used would be equivalent to a human drinking 50 million cups of decaffeinated coffee each day for a lifetime.

3. Other Effects. Exposure to 0.6 percent trichloroethylene was shown to decrease the output of light emitted by the luminous bacteria Photobacterium phosphoreum (65). A general bacteriostatic effect on alpha hemolytic streptococci, gram-positive and gram-negative diplococci, Corynebacterium and Brucella melitensis, was noted in the human oropharyngeal* cavity following exposure to trichloroethylene during anaesthesia (66). E. coli exposed to 1.9 to 3.9 percent trichloroethylene exhibited reduced survival in proportion to the dose, with the higher dose being 100 percent lethal (67).

While trichloroethylene is itself a noncorrosive chemical, it readily undergoes oxidation to acidic materials that are highly corrosive, damaging many metal surfaces with which they come into

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*The oropharynx is the division of the pharynx that lies between the soft palate and upper edge of the epiglottis.

contact.

4. Present, On-going Research (68).

- o "Correlation Between Breathing Zone Solvent Concentrations and Solvent Losses from Vapor Degreasers." N. A. Esmen, University of Delaware, School of Engineering.

A study for the U.S. Department of Defense at Frankford Arsenal is in progress to determine whether or not present ventilation systems offer adequate protection to workers from trichlorcethylene vapor loss. Actual concentrations in the presence and absence of ventilation will be measured and compared to those expected from the particular ventilation system. Causes of overexposure will be identified and recommendations made.

- o "Hepatotoxic Potential of Solvent and Amphetamine Abuse." G. J. Troiger, University of Kansas, School of Pharmacy.

While trichloroethylene in itself does not exhibit significant potential for the production of liver injury, its abuse, via inhalation of cleaning fluids in conjunction with potentiating agents such as acetone (via inhalation of airplane glue) or amphetamines, may represent a potential hazard. This project will evaluate the effects of acetone and amphetamine on chlorinated hydrocarbon hepatotoxicity under conditions similar to those present during abuse. Both adult and immature animals will be tested.

- o "Behavioral/Neurological Evaluation of Solvent Exposures." B. Gutnik and C. Xintaros, U.S. Department of Health, Education and Welfare, P.H.S. Center for Disease Control.

Approximately 150 workers who experience chronic occupational exposure to trichloroethylene will be studied to: (a) detect behav-

ioral and neurological changes; (b) correlate these changes with exposure, body burden, accidents, and illnesses derived from their records; and (c) ascertain the effectiveness of behavioral function measurements as an early warning indicator of adverse exposure.

- "Quick Response Evaluation of Behavioral Effects of Trichloroethylene." C. Xintaros and H. Cohen, U.S. Department of Health, Education and Welfare, P.H.S. Center for Disease Control.

This project will assess the validity of findings reported by Salvini et al. 1971 (31) concerning trichloroethylene effects on human performance capabilities at the current exposure level of 100 ppm. This work is necessary before the Department of Labor establishes a new Trichloroethylene Standard.

C. AMBIENT CONCENTRATIONS, POPULATION AT RISK AND MEASUREMENT TECHNOLOGY

It has been estimated that approximately 600,000 tons of trichloroethylene are released to the atmosphere each year and that 10,000 tons are released to the ocean (64), a combined loss of 610,000 tons per year, or approximately 60 percent of total world production. However, trichloroethylene is not considered to be a persistent environmental contaminant because it has a low water solubility, high vapor pressure, and rapid atmospheric photodegradation rate (sea level half-life of 0.3 days). Using the methodology described in the NAS publication "Assessing Ocean Pollutants" (1975), a transport model for trichloroethylene has been prepared (64). From this analysis, it is concluded that trichloroethylene is leaving the biosphere as rapidly as it is being introduced, and is not accumulating. The short half-life and low concentration suggest that trichloroethylene will not contribute significantly to the levels of chlorine in the stratosphere. Based on data concerning water solubility and vapor pressure and by comparision with similar compounds, CCl_4 and C_2Cl_4, bioconcentration of trichloroethylene should not be a significant environmental factor.

A summary of typical concentrations of trichloroethylene in the various sectors of the environment is presented in Table V. Ambient concentrations in air are about 11 nanograms/m^3, or 20 parts per trillon (ppt). In water, background concentration levels are approximately 0.6 nanograms/liter (0.1 ppt) (64). A background concentra-

TABLE V

OCCURRENCE OF TRICHLOROETHYLENE IN THE ENVIRONMENT
TYPICAL CONCENTRATIONS (W/W)

	MINIMUM	MAXIMUM
Air	10^{-9}	10^{-8}
Rainwater	10^{-11}	10^{-9}
Surface water	10^{-11}	10^{-9}
Potable water	10^{-11}	10^{-9}
Sea water	10^{-10}	10^{-9}
Marine sediments	10^{-10}	10^{-9}
Marine invertebrates	10^{-9}	10^{-8}
Fish	10^{-9}	10^{-8}
Waterbirds	10^{-9}	$> 10^{-7}$
Marine mammals	10^{-9}	$> 10^{-8}$
Fatty foods	10^{-9}	10^{-8}
Non-fatty foods	10^{-9}	10^{-9}
Human organs	10^{-9}	10^{-9}
Human body fat	10^{-9}	10^{-8}

Source: McConnell, G., Ferguson, D.M. and Pearson, C.R. "Chlorinated Hydrocarbons and the Environment." Endeavor 34, 13-18 (1975).

tion in western Ireland of 15 ppt for the period June-July 1975 was reported (69). Areas far removed from urban industrialized centers, however, reported values of less than 5 ppt. Such areas include sites over the North Atlantic (October 1973) (64) and rural Pullman, Washington (December 1974-February 1975) (70).

Ambient levels of trichloroethylene in the vicinity of the production facilities have not been reported. However, it is possible to develop a computed estimate of expected levels.

Five plants in the United States produce trichloroethylene with total annual production of 430 million pounds. Specific details on the operational characteristics and the atmospheric emissions of individual plants are not readily available. It is known, however, that trichloroethylene is emitted from vents, not stacks (71). In order to estimate ambient levels of trichloroethylene in the vicinity of its production units, the use of diffusion equations is necessary. Hypothetical plant conditions could be used as inputs to a Gaussian plume equation* from Turner's Workbook of Atmospheric Dispersion Esti-

*Ground-level downwind concentrations resulting from a point source are predicted by the following equation:

$$\chi(x, 0, 0; H) = \frac{Q}{\pi \sigma_y \sigma_z \mu} \exp\left[-1/2\left(\frac{H}{\sigma_z}\right)^2\right]$$

where: Q = uniform emission rate (grams/sec)
u = mean wind speed affecting the plume (meter/sec)
H = effective stack height; that is, sum of stack height plus plume rise (meters)
σ_y = horizontal dispersion coefficient evaluated in terms of downwind distance to the point for which the concentration is being computed (meters)
σ_z = vertical dispersion coefficient evaluated in terms of downwind distance to the point for which the concentration is being computed (meters)
χ = ground-level concentration on plume axis (grams/meter^3)

mates (72). The basic diffusion equation should be modified, however, due to the effect of the plant itself on the flow of air. Mechanical turbulence in the wake of a building tends to produce aerodynamic downwash, resulting in the fairly rapid diffusion of a gaseous emission down to the ground. This region of disturbed flow extends downwind a distance equal to several times the height of the building. While this situation does not result in a Gaussian distributed plume, a modification of the formula still can be employed. This is because the turbulent mixing in the wake of the building is assumed to be distributed uniformly in the vertical direction, analogous to the situation where a plume is trapped below an inversion layer. A limited vertical mixing height can be modeled using the equation:

$$\chi(x, 0, z; H) = \frac{Q}{\pi\sigma_y u\ (.8L)}$$

where L is the limit of the mixing depth and z is $\leq$ L. In the case of an isolated rectangular building, it is assumed that L equals 1.5 times the building height.

In addition, there is a horizontal wind turbulence which is assumed to result in an initial horizontal plume spread equal to the width of the building normal to the wind direction. This is analogous to an area source emission where the area source is the building top. This can be modeled using a further modification of the basic Gaussian diffusion equation. A virtual point source is assumed upwind from the building at a distance where the plume spread (for

the given stability conditions) would equal the crosswind width of the building. The modified diffusion equation is therefore:

$$\chi(x, 0, z; H) = \frac{Q}{\pi\sigma'_y u\ (1.2h)}$$

where H is the building height and σ'_y is based on the downwind distance to the receptor point plus the upwind distance to the vertical point source.

Assuming that a typical plant producing trichloroethylene is equipped with control equipment having a 98 percent removal efficiency, a plant producing 280 million pounds of trichloroethylene annually would have a potential trichloroethylene emission rate of 80 grams per second. With reasonable assumptions, the concentration 500 meters downwind of a plant would be approximately 570 $\mu g/m^3$, or 0.106 ppm.* An individual standing 500 meters downwind of a production facility would have to remain there for 11.4 years in order to inhale the minimum amount of trichloroethylene reported to result in slight central nervous system depression (5 ppm, 40 hours/week for 50 weeks). He must remain for 228 years in order to inhale an amount equivalent to that inhaled by an industrial worker occupationally exposed to 100 ppm over an 8-hour work day.

Several studies have been performed on atmospheric concentrations of trichloroethylene in vapor degreasing workshops where the

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*Assumptions employed are: (1) trichloroethylene is nonreactive; (2) atmospheric stability class is neutral; (3) wind speed is 6 meters/sec.; (4) building height is 50 feet (15.24 meters); and (5) width is 100 feet (30.48 meters).

major danger of exposure exists. In one study, Grandjean (73) measured trichloroethylene concentrations in the air surrounding cold trichloroethylene vats wholly lacking in safety devices or ventilation systems. Measured values ranged from 67 to 157 ppm (average, 105 ppm) trichloroethylene. While vapor degreasing operations were being performed, the average concentration in the air was 167 ppm. Installation of appropriate ventilation and exhaust systems decreased the concentration to 53 ppm. Atmospheric concentrations during a twice-weekly cleaning procedure were 1120 ppm outside the tank as emptying took place and 815 ppm inside the tank for the duration of the 30-minute cleaning operation.

Ahlmark et al. (74) performed an extensive study of 18 degreasing tanks in 14 plants in Sweden. The extent of exposure was determined in terms of time and concentrations by sampling breathing zones for a variety of operations. Mean exposure was 50 ppm (range 0 to 400 ppm) for degreasing operations, while the highest mean value was 225 ppm (range 10 to 375 ppm) for the cleaning operation. Results of this study are summarized in Table VI.

The National Institute of Occupational Health and Safety has estimated that approximately 200,000 workers are exposed to trichloroethelene in the United States (1). No detailed statistics were provided. It was estimated, however, that the majority of exposed workers are employed in industry associated with vapor degreasing of metals. The smaller number of workers also exposed are primarily

TABLE VI

TRICHLOROETHYLENE EXPOSURE IN SWEDISH ENGINEERING WORKSHOPS*

Job Location	Minutes**	Trichloroethylene (ppm) Range	Mean
Open air	2.5	0-0	0
Less than 5 meters from tank	4.8	0-140	23
Greater than 5 meters from tank	4.5	0-80	7
Sorting, before degreasing	2.7	0-375	32
Degreasing	4.7	0-400	50
Sorting, after degreasing	2.4	0-375	46
Cleansing	4.8	10-375	46
Inspection of tank	4.7	0-375	70
Refilling	4.8	10-415	36

*Ahlmark, A., Gerhardsson, G. and Holm, A. "Trichloroethylene Exposure in Swedish Engineering Workshops" in Proceedings of the XIVth Internation Congress on Occupational Health, Madrid, Sept. 1963, pp.448-450.

**Mean time spent by each operator at the various locations. It is understood that an operator may perform at a given location with greater frequency than another operator.

employed in industries which consume trichloroethylene (e.g., dry cleaning plants). A negligible number of workers is believed to be exposed at production facilities. The main exposure hazard is for the users, not the producers, of trichloroethylene, since the chemical is synthesized in a closed-system apparatus. Dow Chemical and PPG Industries, the two largest producers of trichloroethylene in the United States, claim that levels in their production plants are well below 100 ppm (63). A Dow spokesman noted that, in contrast, the average level in degreasing plants is usually below 100 ppm, but considerable levels above this limit are common (63). In 1973, OSHA recommended a 100 ppm ceiling in the air as a time-weighted average during an 8-hour workday. They also recommended a 200 ppm maximum for 15 minute's exposure and a 300 ppm upper limit (1).

Dow and PPG claim that none of their trichloroethylene customers use this chemical in a consumer product (73). However, it is found in products sold directly to the public. A home cleaning fluid found on the shelf of a suburban Washington, D.C. drugstore was labeled as containing trichloroethylene (63). And since trichloroethylene is commonly used to extract caffein from coffee and oleoresin flavorings from spices, ingestion of this substance by the public does occur. Current FDA limits are 10 ppm of trichloroethylene in decaffeinated instant coffee, 25 ppm in decaffeinated ground coffee; and 30 ppm in spices (63).

Determination of concentrations of trichloroethylene in air

samples is a two-step process, requiring collection and analysis. Collection methods include evacuated gas sampling flasks (75), plastic bags, absorbents (79), and activated charcoal (80-82). Three types of analytical methods are available: physical methods, such as interferometry or gas chromatography; chemical methods, which depend upon a specific reaction of trichloroethylene; and destructive methods, such as combustion techniques that decompose trichloroethylene and liberate hydrochloric acid (76). Field methods for estimation of trychloroethylene vapor in the air include use of the Davis Halide Meter and direct reading colorimetric indicator tubes (83, 84).

Adsorption onto activated charcoal represents the easiest and most efficient method of collection. Utilization of adsorbing liquids is inconvenient, especially for collecting breathing-zone samples. Use of plastic bags or evacuated containers often results in loss of sample due to adsorption onto the walls, permeation through the plastic, or escape around joints. On humid days, the excessive water vapor tends to displace trichloroethylene from silica gel when this method is employed (85).

Gas chromatography is the most specific and sensitive of all the measurement methods in use and is the method of choice (81, 85-87). Other methods are subject to inaccuracy due to interferences by other chlorinated solvents or chloride-containing compounds, and are acceptable only if trichloroethylene is the sole contaminent (78, 83).

Biologic monitoring of the exposed workers is also possible. A preferred method consists of breath analysis--that is, determination of the trichloroethylene concentration in the exhaled air of a given worker. It has been determined that a breath sample collected approximately three hours after exposure will provide a concentration of trichloroethylene that is directly proportional to the time-weighted average concentration experienced during exposure (30).

D. SOURCES

Trichloroethylene was first prepared by Fischer in 1864 in the course of experiments on the reduction of hexachloroethane with hydrogen. A process for the manufacture of trichloroethylene from acetylene was developed in Vienna between 1903 and 1905, and commercial production commenced in 1908. Trichloroethylene was not produced commercially in the United States until 1925. At that time, only a few hundred tons per year were consumed in minor extraction processes and in such products as boot polish and printers' ink driers. It was not until the 1930's that the applications in metal degreasing and dry cleaning operations placed trichloroethylene in strong demand (?).

Before 1967, 85 percent of the trichloroethylene produced in the United States was prepared from acetylene by chlorination to form 1,1,2,2-tetrachloroethene, followed by dehydrochlorination of the latter to the desired product. At present, 85 percent of trichloroethylene production proceeds via the chlorination or oxyhydrochlorination of ethylene, with the intermediate formation of ethylene dichloride. The latter is then further chlorinated to trichloroethylene (1).

There are five producers of trichloroethylene in the United States today. The locations of their plants and their production capacities are listed in Table VII. A map of the production facilities is given in Figure 1. Trichloroethylene is also produced in

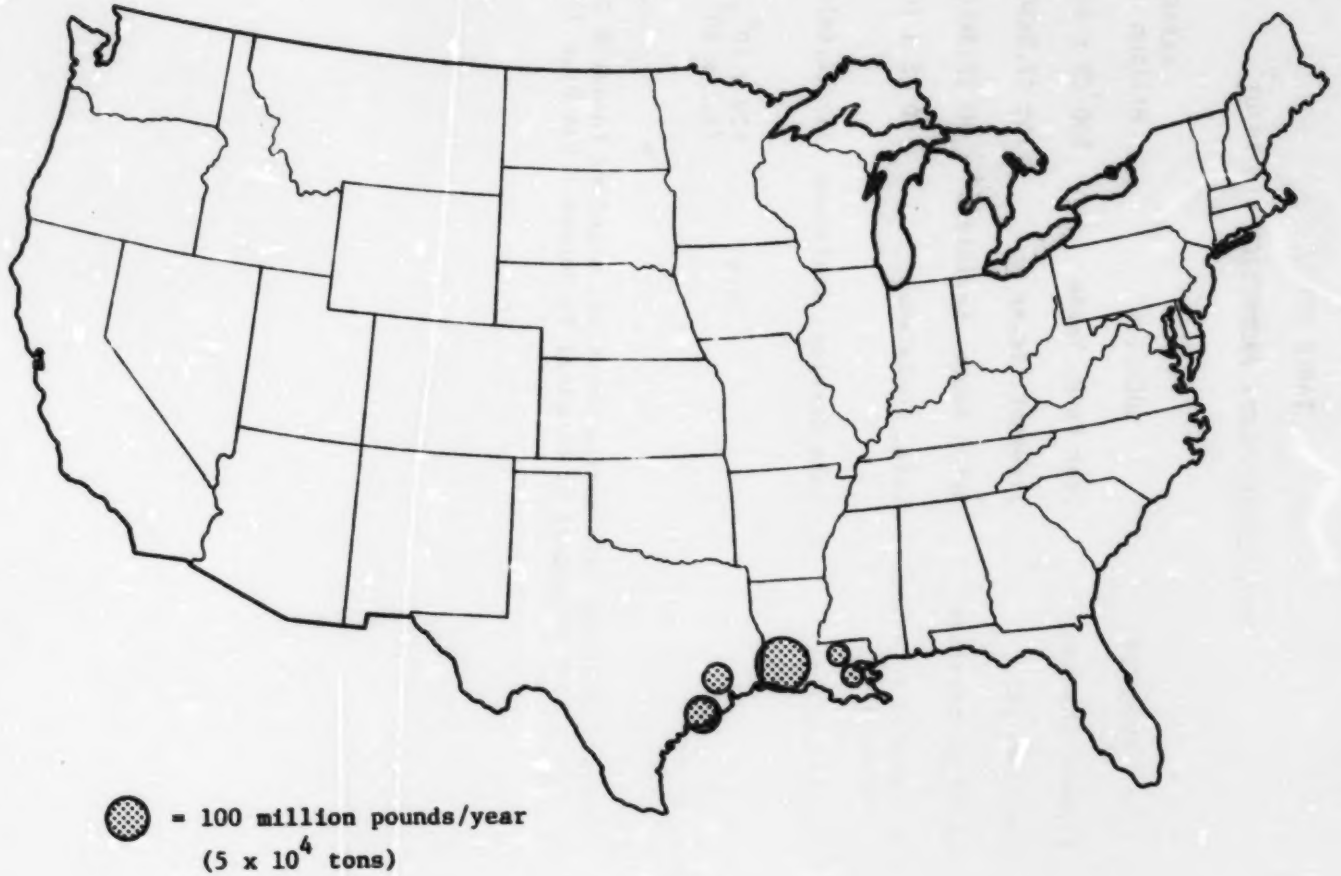
TABLE VII

TRICHLOROETHYLENE PRODUCTION (in 1970)[1]

PRODUCER	LOCATION	CAPACITY Million Lbs./Yr.
Diamond Shamrock	Deer Park, Texas	100 (5×10^4 tons)
Dow Chemical	Freeport, Texas	150 (7.5×10^4 tons)
Ethyl Corporation	Baton Rouge, Louisiana	50 (2.5×10^4 tons)
Occidental Petroleum (Hooker)	Taft, Louisiana	40 (2×10^4 tons)
PPG Industries	Lake Charles, Louisiana	280 (1.4×10^5 tons)
	Total	620×10^6 lbs. (3.1×10^5 tons)

Source: Chemical Information Services, Stanford Research Institute. 1975 Directory of Chemical Producers - USA Page 725.

FIGURE 1
TRICHLOROETHYLENE PRODUCTION CAPACITY



many countries around the world and a significant amount has been imported into the United States over the years.

The annual peak production level of trichloroethylene in the United States occurred in 1970. During that year, 610 million pounds (305,000 tons) were manufactured. In 1966, the classification of trichloroethylene as a photochemically reactive smog contributant resulted in a decreased use of the chemical, which was subsequently reflected in production figures. At present, about 430 million pounds (215,000 tons) are manufactured in the United States (63). The world annual production capacity is estimated (1973) to be approximately one million tons (62).

Trichloroethylene is produced by feeding oxygen-free high-purity chlorine gas and acetylene into a packed tower at 50°C and atmospheric pressure. The tower is equipped with a condenser to reflux part of the tetrachloroethane product. Chlorine and acetylene react to form tetrachloroethane, which is partially withdrawn from the reactor, vaporized, and superheated to 300°C. It is then fed to a tower filled with activated carbon (dehydrochlorination), where trichloroethylene is formed. Hydrogen chloride, trichloroethylene, and unconverted tetrachloroethane from the dehydrochlorination are fed to a stripping column where the hydrogen chloride is stripped off overhead and absorbed in water as hydrochloric acid. Bottoms are fed to a second stripper where trichloroethylene is taken off overhead. This process for producing trichloroethylene has two air

pollution emission points, the vent on the reflux condenser connected to the reactor and the vent on the tail gas absorber (71).

Over 90 percent of all trichloroethylene produced is used by industrial metal fabricating plants for vapor degreasing. Since the late 1960's, tetrachloroethylene and methylene chloride have also been used for this purpose. However, trichloroethylene still commands 50 percent of the vapor degreasing market. The remaining 5 to 10 percent of the trichloroethylene produced is utilized for a wide variety of applications (2). As an extractive solvent, trichloroethylene has been used for the selective extraction of foods and medicines, including fish meal, meat meal, oil-containing seeds, soya beans, coffee beans (for decaffeination), spices, and alkaloids. Analyses of a wide range of foods of both anima. and vegetable origin for trichloroethylene residues have been performed. The results are presented in Table VIII. General Foods, Inc., the largest producer of decaffeinated coffee, has recently disclosed that it is abandoning the use of trichloroethylene for decaffeinating coffee. Instead, it is replacing this solvent with methylene chloride (88). As a dry cleaning solvent, trichloroethylene has been used for removing oils and waxes from both natural fibers (cotton and wool) and synthetic fibers (rayon, fiberglass, and cotton polyester material) by the textile industry. Other minor applications include: fur cleaning agent, motion picture film cleaner, dilutent or carrier in paints, adhesives and varnishes, formulation in paint removers, low tempera-

TABLE VIII

TRICHLOROETHYLENE IN FOODSTUFFS
(CONCENTRATIONS IN µg/kg)

FOODSTUFF	TRICHLOROETHYLENE
Dairy Produce:	
Fresh milk	0.3
Cheshire cheese	3
English butter	10
Hen's eggs	0.6
Meat:	
English beef (steak)	16
English beef (fat)	12
Pig's liver	22
Oils and fats:	
Margarine	6
Olive oil (Spanish)	9
Cod liver oil	19
Vegetable cooking oil	7
Castor oil	ND
Beverages:	
Canned fruit drink	5
Light ale	0.7
Canned orange juice	ND
Instant coffee	4
Tea (packet)	60
Wine (Yugoslav)	0.02
Fruit and vegetables:	
Potatoes (S.Wales)	ND
Potatoes (N.W. England)	3
Apples	5
Pears	4
Tomatoes*	1.7
Black grapes (imported)	2.9
Fresh bread	7

*Tomato plants were grown on a reclaimed lagoon at Runcorn Works of ICI.

ND - Not detected.

Source: McConnell, G., Ferguson, D.M. and Pearson, C.R. "Chlorinated Hydrocarbons and the Environment." Endeavor 34, 13-18 (1975)

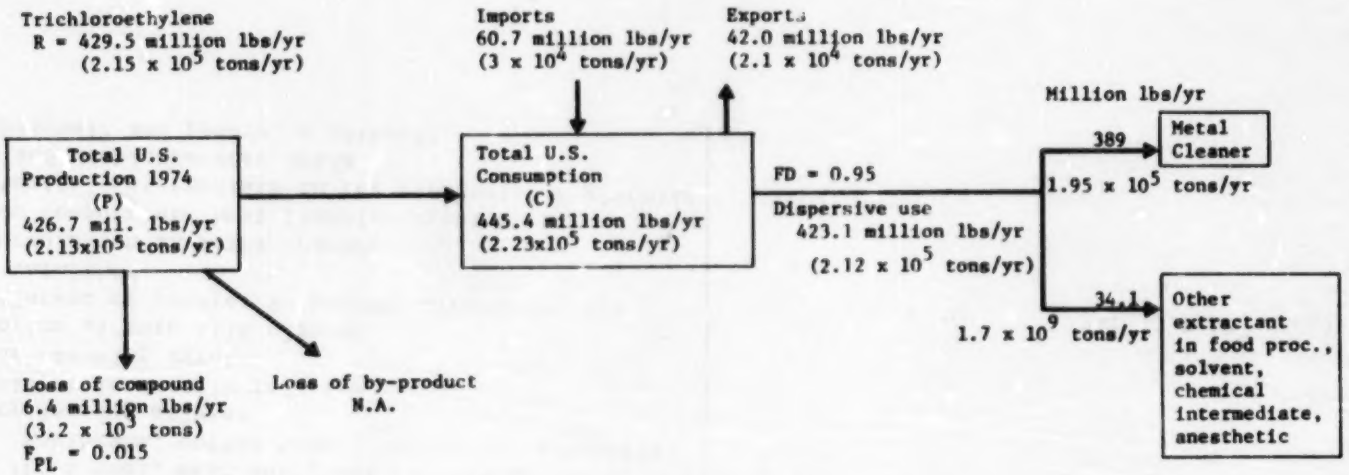
ture heat transfer agent, and chemical intermediate in the synthesis of glycolic and dichloroacetic acids. Trichloroethylene also experiences limited use as an analgesic and general anaesthetic, with about 60,000 patient exposures per year (63). Industrial uses are summarized in Table IX and a flow chart of distribution is presented in Figure 2.

TABLE IX

INDUSTRIAL OR END USE OF TRICHLOROETHYLENE (1974)

USE	PERCENT	AMOUNT (Millions Lbs.)
Vapor degreasing of metals	> 90	389 (1.95×10^5 tons)
Food and drug extraction (fish meal, meat meal, oil containing seeds, soyabeans, coffee beans, spices and alkaloids) Drycleaning solvent Solvent in textile industry Fur cleaning agent Motion picture film cleaner Dilutent or carrier in paints, adhesives, and varnishes Formulation in paint remover Low temperature heat transfer agent Chemical intermediate in the synthesis of glycolic and dichloroacetic acids Analgesic and general anesthetic	< 10	341 (1.7×10^4 tons)

FIGURE 2
SUMMARY OF PRODUCT RELEASE INFORMATION



Source: S.L., Chan, F.Y., Jones, J.L., Liu, D.H., McCaleb, K.E., M.N.T., Sopios, K.N. and Schendel, D. E. "Research Program on Hazard Priority Ranking of Manufactured Chemicals." SRI Project ECU-3386. Stanford Research Institute, Menlo Park, California.

R = production rate

F_D = fraction dispersal

F_{PL} = fraction of production lost

E. CONTROL

Workers in industrial or commercial operations utilizing trichloroethylene run the major risk of exposure. Therefore, it is essential that careful controls be implemented such that the time-weighted average concentration in the atmosphere over an 8-hour work day for these personnel does not exceed 100 ppm. Several methods are available to control the emission of solvent vapors into the general work environment (1).

1. Substitution of Less Harmful Solvents. Any solvent considered a potential replacement for trichloroethylene must first be examined in terms of effectiveness, relative volatility, volume of air required to dilute the vapor in the work environment to a safe level, relative amounts of each solvent required for the task, likelihood of control of vapor concentration, and flammability. Tetrachloroethylene and dichloroethylene have been found effective in both degreasing and dry cleaning applications (63, 2).

2. Proper Design of Condensation Apparatus. High concentrations of trichloroethylene in the atmosphere, especially around degreasing operations, are a result of solvent escape from evaporation and spills. It is, therefore, important to control the evaporation and condensation rates by an appropriate balance between heat input and condensation capacity of refrigerated condenser coils.

3. Process Ventilation and Process Location. Ventilation is by far the most common engineering method for controlling solvent

vapors and vapor recovery systems are possible when a high airflow exhaust system is required for good ventilation. Grandjean (73) investigated the effectiveness of a fixed lateral exhaust system, intended to remove rising trichloroethylene vapor at the top of a degreasing tank. The system lowered the average trichloroethylene concentration from 167 to 112 ppm. In one case where both an exhaust system and a vapor condensation system were operating, the average concentration of atmospheric trichloroethylene was reduced from 105 to 30 ppm.

A further decrease in the concentration of trichloroethylene vapors can be expected if the degreasing operation is located in a large room with good ventilation wherein the entire work area is continuously flushed with uncontaminated air. If the exhaust system is properly designed, it will prevent the accumulation or recirculation of trichloroethylene vapors in the operation area. In the Grandjean study (73), addition of an air-blowing system to improve general ventilation further lowered the trichloroethylene concentration from 112 to 53 ppm. Areas in the vicinity of doors, windows, or other possible sources of draft conditions should be avoided since excessive air movements could decrease the effectiveness of local exhaust systems. Skinner (89) reported the use of baffles on windows near degreasers to control high velocity drafts. This procedure reduced the trichloroethylene concentration from a range of 170 to 230 ppm to 30 to 40 ppm.

Finally, trichloroethylene vapors should be kept away from high energy-high temperature sources to reduce the possibility of decomposition into chlorine, hydrogen chloride, or phosgene.

4. Proper Effluent Treatment. Scrubbers used on packed towers, such as those used for the production of trichloroethylene, have control efficiencies ranging from 90 percent to close to 100 percent (90, 91).

5. Proper Handling, Maintenance, and Disposal Procedures. In small quantities, trichloroethylene should be stored and transported in lined tins or galvanized mild steel drums fitted with screwcaps or other suitably tight tops. Small quantities may also be stored in amber or green-colored glass containers. Large quantities should be stored in mild steel tanks fitted with a drier to prevent the ingress of moist air. Ventilation should lead to a safe point outside the storage area (2).

Immediate cleanup of spills, periodic inspection of equipment, and immediate repair of damaged equipment and leaks must be practiced to minimize both atmospheric contamination and accidental skin contact with trichloroethylene. Waste materials from cleaning that are contaminated should be stored in a well-ventilated area until ready for disposal, and eventually incinerated. Small quantities of trichloroethylene should be poured into a mixture of 10 percent soda ash and sand, placed in a suitable container, and incinerated (1).

Since a wide spectrum of adverse effects, ranging from head-

aches to fatalities, have been reported subsequent to trichloroethylene exposure, it is essential that concentrations to which workers are exposed remain below the presently established "no effects" level of 100 ppm. Thus, effective monitoring programs must be established. Breathing-zone samples should be collected frequently for each type of job in which exposure is suspected. The sample should be collected by drawing air through a charcoal tube to trap the organic vapors and extraction of the trapped sample may be accomplished with carbon disulfide. An aliquot may then be injected into a gas chromatograph and trichloroethylene concentration determined (60).

REFERENCES

1. U.S. Department of Health Education and Welfare, "Criteria for a recommended standard--occupational exposure to trichloroethylene." 1973.

2. Hardie, D. W. F., "Trichloroethylene," Encyclopedia of Chemical Technology V. 5, Interscience, 1964.

3. Irish, D. D., "Trichloroethylene," Industrial Hygiene and Toxicology, second revised edition, Vol II Toxicology, Frank A. Patty, editor, 1976.

4. National Science Foundation Panel on Manufactured Organic Chemicals in the Environment, SRI data, 1975.

5. Barrett, H. M., and Johnston, J. H., "The Fate of trichloroethylene in the organism," J. Biol Chem. 127, 765-70, 1939.

6. Powell, J. F., "Trichloroethylene: Absorption, elimination and metabolism," Brit. J. Ind. Med. 2, 142-45, 1945.

7. Butler, T. C., "Metabolic transformations of trichloroethylene," J. Pharmacol. Exp. Ther. 97, 84-92, 1949.

8. Souek, B., and Vlachova, D., "Excretion of trichloroethylene metabolities in human urine," Brit. J. Ind. Med. 17, 60-64, 1960.

9. Bartoniek, V., "Metabolism and excretion of trichloroethylene after inhalation by human subjects," Brit. J. Ind. Med. 19, 134-41, 1962.

10. Ogata, M., Takatsuka, Y. and Tomokuni, K., "Excretion of organic chlorine compounds in the urine of persons exposed to vapours of trichloroethylene and tetrachloroethylene." Brit. J. Ind. Med. 28, 386-91, 1971.

11. Ikeda, M., Ohtsuji, H., Imamura, T., and Komoike, Y., "Urinary excretion of total trichloro-compounds, trichloroethanol and trichloro acetic acid as a measure of exposure to trichloroethylene and tetrachloroethylene," Brit. J. Ind. Med. 29, 328-33, 1972.

12. Lilis, R., Stanescu, D., Muico, N., and Roventa, A., "Chronic effects of trichloroethylene exposure," Med. Lav. 60, 595-601, 1969.

13. Andersson, A., "Health dangers in industry from exposure to trichloroethylene," Acta Med. Scan (Sup 323) 157, 7-220, 1957.

14. Bardodej, Z., and Vyskocil, J., "The problem of trichloroethylene in occupational medicine," Arch. Ind. Health 13, 581-92, 1956.

15. Maloof, C. C., "Burns of the skin produced by trichloroethylene vapors at room temperature," J. Ind. Hyg. Toxicol 31, 295-96, 1949.

16. McBirney, R. S., "Trichloroethylene and dichloroethylene poisoning," Arch. Ind. Hyg. Occup. Med. 10, 130-33, 1954.

17. Reinl, W., "Scleroderma under the influence of trichloroethylene?" Zentrolbl. Arbeits. Med. 7, 58-60, 1957.

18. Stewart, R. D., and Dodd, H. C. "Absorption of carbon tetachloride, trichloroethylene, tetrachloroethylene, methylene chloride and 1,1,1 - trichloroethane through the human skin," Am Ind. Hyg. Assoc. J. 25, 439-46, 1964.

19. Ahlmark, A., and Forssman, S., "Evaluating trichloroethylene exposures by urinalyses for trichloroacetic acid," Arch. Ind. Hyg. Occup. Med. 3, 386-98, 1951.

20. Frant, R., and Westendorp, J., "Medical control on exposure of industrial workers to trichloroethylene," Arch. Ind. Hyg. Occup. Med. 1, 308-18, 1950.

21. Friberg, L., Kylin, B. and Nystrom, A., "Toxicities of trichloroehtylene and tetrachloroethylene and Fujiwara's pyridine - alkali reaction," Acta Pharmacol. Toxicol. 9, 303-12, 1952.

22. Quadland, H. P., "Petroleum solvents and trichloroethylene"--Part 3 of the literature study of reports of occupational diseases attributed to volatile solvents, Ind. Med. 13, 45-50, 1944.

23. Kunz, E., and Isenschmidt, R., "The toxic effect of trichloroethylene on the eye," Klin. Monatsb. Augenheillkd 94, 577-85, 1935.

24. Friborska, A., "The phosphatases of peripheral white blood cells in workers exposed to trichloroethylene and perchloroethylene," Brit. J. Ind. Med. 26, 159-61, 1969.

25. Albaha C., Guyotjeannin, C., Flaisler, A., and Thiaucourt, P., "Transaminases and occupational exposure to trichloroethylene," Arch. Mol. Prof., 20, 421-46, 1959.

26. Talot, F., Viallier, J., Roullett, A., Rivoire, J., and Figueres, J. C., "Hepatic toxicity of trichloroethylene," Arch. Mol. Prof. 25, 9-15, 1964.

27. Milby, T. H., "Chronic trichloroethylene intoxication," J. Occup. Med. 10, 252-54, 1968.

28. Gyotjeannin, C., and Van Steenkiste, J., "Action of trichloroethylene on proteins and serum lipids--study of 18 employees working in a contaminated atmosphere," Arch. Mal. Prof. 19 , 489-94, 1958.

29. Vernon, R. J., and Ferguson, R. K., "Effects of trichloroethylene on visual-motor performance," Arch. Environ. Health. 18, 894-900, 1969.

30. Stewart, R. D., Dodd, H. C., Gay, H. H., Erley, D. S., "Experimental human exposure to trichloroethylene," Arch. Environ, Health 20, 64-71, 1970.

31. Salvini, M., Binaschi, S., and Riva, M., "Evaluation of the phsychophysiological functions in humans exposed to trichloroethylene." Br. J. Ind. Med. 28, 293-95, 1971.

32. Kleinfeld, M., and Tabershaw, I. R., "Trichloroethylene toxicity--Report of five fatal cases," Arch. Ind. Hyg. Occup. Med. 10, 134-41, 1954.

33. Secchi, G.C., Chiappino, G. Lotto, A. and Zurlo, N., "Actual chemical composition of the commercial trichloroethylenes and their liver toxicity. Clinical and enzymological studies," Med. Lav. 59, 486-97, 1968.

34. James, W. R. L., "Fatal addiction to trichloroethylene," Brit. J. Ind. Med. 20, 47-49, 1963.

35. St. Hill, C. A., "Occupation as a cause of sudden death," Trans. Soc. Occup. Med. 16, 6-9, 1966.

36. Bell, A., "Death from trichloroethylene in a dry cleaning establishment," N.Z. Med. J. 50, 119-26, 1951.

37. Joron, G. E., Cameron, D. G., and Halpenny, G. W., "Massive necrosis of the liver due to trichloroethylene," Con. Med. Ass. J. 73, 890-91, 1955.

38. Mitchell, A. B. S., and Parsons-Smith, B. G., "Trichloroethylene neuropathy," Br. Med. J. 1, 422-23, 1969.

39. Tomasini, M., and Sartorelli, E., "Chronic intoxication from commercial trielin inhalation with compromise of the eig th cranial nerves," Med. Lav. 62, 277-80, 1971.

40. Todd, J., "Trichloroethylene poisoning with paranoid psychosis and lilliputian hallucinations," Br. Med. J. 7, 439-40, 1954.

41. Baronicek, V., and Soucek, B., "The metabolism of trichloroethylene in rabbits," Arch. Gerverbepator Gerverbehyg. 17, 283-93, 1954.

42. Fabre, R., and Truhaut, R., "Contribution to the study of trichloroethylene toxicology--II. Results of experimental studies in animals," Brit. J. Ind. Med. 9, 39-43, 1952.

43. Adams, E. M., Spencer, H. C., Rowe, V. K., McCollister, D. D., and Irish, D. D., "Vapor toxicity of trichloroethylene determined by experiments on laboratory animals," Arch. Ind. Hyg. Occup. Med. 4, 469-81, 1951.

44. Prendergast, J. A., Jones, R. A.; Jenkins, L. J. Jr.; and Siegel, J., Effects on experimental animals of long-term inhalation of trichloroethylene, carbon tetrachloride, 1,1,1 - trichloroethane, dichlorodifluoromethane and 1,1 - dichloroethylene." Toxicol. Appl. Pharmacol 10:270-89, 1967.

45. Cagianelli, M. A., Menchini, G., Menchini, G. F., and Bianchi, F., "Histological and humoral aspects of chronic experimental trichloroethylene intoxicantions," Boll. Soc. Med. - Chir Pisa 33, 558-70, (1965).

46. Wirtschafter, Z. T., and Cronyn, M. W., "Relative hepatotoxicity--pentane, trichloroethylene, benzene and carbon tetrachloride." Arch. Environ Health 9, 180-85, 1964.

47. Barrett, H. M., Mackean, D. L., and Cunningham, J. G., "A comparison of the toxicity of carbon tetrachloride and trichloroethylene." J. Ind. Hyg. & Toxicol. 20, 360-79, 1938.

48. Seifter, J., "Liver injury in dogs exposed to trichloroethylene." J. Ind. Hyg. Toxicol 26, 250-52, 1944.

49. Kylin, B., Sumegi, I., and Yllner, S., "Hepatotoxicity of inhaled trichloroethylene and tetrachloroethylene -- long term exposure," Acta Pharmacol. Toxicol. 22, 379-85, 1965.

50. Mosinger, M., and Fiorentini, H., "Hepatic, renal, lymphatic and splenic reactions in experimental trichloroethylene intoxication in cats," Compt. rend. soc. biol. 149, 150-52, 1955.

51. Viallier, J., and Casanova, F., "Transaminase levels in the blood of trichloroethylene-treated rats and rabbits," Compt. Rend. Soc. Biol. 159, 221]-21, 1965.

52. Savic, S. M., and Baaske, H., "Biochemical changes in rabbit eyes in trichloroethylene poisoning," Albrecht von Graefes Arch. Kin. Exp. Ophthalmol. 175, 1-6, 1968.

53. Zadorozhnyi, B. V., "Changes in animals in the case of chronic inhalation of trichloroethylene in low concentrations," Gig. Tr. Prof. Zabol. 17, 55-57, 1973.

54. Shmuter, L. M., "Effect of chronic action of small concentrations of chlorinated hydrocarbons on the production of various classes of immunoglogulins," Gig Sonit 37, 36-40, 1972.

55. Mikiskora, H., Mikiska, A., "Trichloroethanol in trichloroethylene poisoning," Brit. J. Ind. Med. 23, 116-25, 1966.

56. Desoille, H., Pinchon, R. A., Lille, F., and Bourguignon, A., "Sequelae of acute intoxication by solvents--Importance of electroencepholography," Arch. Mol. Prof. 23, 5-17, 1962.

57. Desoille, H., Pinchon, R. A., Jons, M., and Bourguigon, A., "Acute experimental trichloroethylene intoxication--aggravating effects of associated chronic alcoholism," Arch. Mol. Prof. 23, 653-64, 1962.

58. Battig, K., and Grandjean, E., "Chronic effects of trichloroethylene on rat behavior--Effects on swimming performance, exploratory behavior, and maze and avoidance learning," Arch. Environ. Health 7, 694-99, 1963.

59. Grandjean, E., "The effects of short exposures to trichloroethylene on swimming performances and motor activity of rats," Am. Ind. Hyg. Assoc. J 24, 376-79, 1963.

60. Carpenter, C. P., Smyth, H. F. Jr., and Pozzani, O. C., "The assay of acute vapor toxicity, and the grading and interpretation of results on ninety-six chemical compounds," J. Ind. Hyg. Toxicol. 31, 343-346 (1949).

61. Bernardi, L., Penzani, B., and Luvoni, R., "Histologic researches on the rabbit brain in trichloroethylene intoxication," Ras. Med. Ind. 25, 269-276, 1956.

62. McConnell, G., Ferguson, D. M., and Pearson, C. R., "Clorinated Hydrocarbons and the Environment" Endeavor 34, 13-18 (1975).

63. Elkins, H. B., Holby, A. K., and Fuller, J. E., "The determination of atmospheric contaminants--I. Organic halogen compounds," J. Ind. Hyg. Toxicol. 19, 474-85, 1937.

64. Lugg, G. A., "Fujiwara reaction and determination of carbon tetrachloride, chloroform, tetrachloroethane and trichloroethylene in air," Med. J. Aust. 38, 1532-36, 1966.

65. Campbell, E. E., Milligan, M. F., and Miller, H. M., "Evaluation of methods for the determination of halogenated hydrocarbons in air," Am. Ind. Hyg. Ass. J. 20, 138-41, 1959.

66. Peterson, J. E., Hoyle, H. R., and Schneider, E. J., "The analysis of air for halogenated hydrocarbon contaminants by means of absorption on silica gel," Am. Ind. Hyg. Assoc. Q 17, 429-33, 1956.

67. Cook, W. A., and Coleman, A. L., "Determination of injurious constituents in industrial atmospheres--II. Determination of solvent vapors in air by means of activated charcoal," J. Ind. Hyg. Toxicol. 18, 194-210, 1936.

68. Reid, F. H., and Halpin, W. R., "Determination of halogenated and aromatic hydrocarbons in air by charcoal tube and gas chromotography," Am. Ind. Hyg. Assoc. J. 29, 390-96, 1968.

69. Kupel, R. E., and White, L. D., "Report on a modified charcoal tube," Am. Ind. Hyg. Ass. J. 32, 456, 1971.

70. Schaffer, A. W., and Hoyle, H. R., "Nine years' experience with the Davis Halide meter," Am. Ind. Hyg. Assoc. J. 22, 93-96, 1961.

71. Saltzman, B., "Direct reading colorimetric indicators," American Conference of Governmental Industrial Hygienists: Air Sampling Instruments for Evaluation of Atmospheric Contaminants, fourth edition, Cincinnati, Ohio, pp 51-510 & 528, 1972.

72. Whitman, N. E., and Johnston, A. E., "Sampling and analysis of aromatic hydrocarbon vapors in air: A gas-liquid chromatographic method," Am. Ind. Hyg. Assoc. J. 25, 464-69, 1964.

73. White, L. D., Taylor, D. G., Mauer, P. A., and Kupel, R. E., "A Convenient optimized method for the analysis of selected solvent vapors in the industrial atmosphere," Am. Ind. Hyg. Ass. J. 31, 225-32, 1970.

74. Urone, P., and Smith, J. E., "Analysis of chlorinated hydrocarbons with the gas chromatograph," Am. Ind. Hyg. Assoc. J. 22, 36-41, 1961.

75. Seltzer, R. J., "Reactions Grow to Trichloroethylene Alert," Chemical and Engineering News, 41-43, May 19, 1975.

76. Moolenar, R. (Dow Chemical Company), personal communication, July 1975.

77. White, D. C., and Dundas, C. R., "Effect of anesthetics on emission of light by luminous bacteria," Nature 226, 456-8, 1970.

78. Musinu, C., Caboni, F., and Pittoni, L., "Effect of some halogenated anesthetics on the orotracheal flora," Minerva Anestesiol. 35, 1021-3, 1969.

79. Horton, J. N., Sussman, M., and Mushin, W. W., "Antibacterial action of anesthetic vapors," Brit. J. Anaesth. 42, 483-7, 1970.

80. Smithsonian Science Information Exchange.

81. Lovelock, J. E., "Atmospheric halocarbons and stratospheric ozone," Nature 252, 292-294, 1974.

82. Grimsrud, E. P., and Rasmussen, R. A., "Survey and analysis of halocarbons in the atmosphere by gas chromatography-mass spectrometry," (1975) in preparation. (Personal communication from R. Moolenar, Dow Chemical Company, July 1975).

83. Sittig, M., Pollution Control in the Organic Chemical Industry, 208, (1974).

84. Turner, D. G., Workbook of Atmospheric Dispersion Estimates, U.S. Department of Health, Education and Welfare, Revised, 1969.

85. Grandjean, E., Munchinger, R., Turrian, V., Haas, P. A., Knoepfel, H. K., and Rosenmund, H., "Investigation into the effects of exposure to trichloroethylene in mechanical engineering," Br. J. Ind. Med 12, 131-42, 1955.

86. Ahlmark, A., Gerhardsson, G., and Holm, A., "Trichloroethylene exposure in Swedish engineering workshops," Proceedings of the XIVth International Congress on Occupational Health, Madrid, September 1963, pp 448-50.

87. Stock, V. T., Jr., Forrest, D. E., and Wahl, K. K., "Determination of trichloroethylene in air," Am. Ind. Hyg. Assoc. J. 22, 184-86, 1961.

88. Anonymous, Washington Post, July 17, 1975.

89. Skinner, J. B., "Control of health hazards in the operation of metal degreasers," Am. Ind. Hyg. Assoc. Quart 13, 11-16, 1952.

90. Kempner, S. K., E. N. Seiler, and D. H. Bowman, "Performance of commercially available equipment in scrubbing hydrogen chlorid gas," J. of the Air Pollution Control Association, Volume 20, No. 3, pg. 139-143.

91. Dyer, J., and J. W. Wett, "Scrubber collects HCl and other pollutants," Chemical Processing, January 1969.

92. U.S. Dept. of Health Education and Welfare, J. A. Danielson, editor--Air Pollution Engineering Manual, 1967.